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Case Presentation

Sciatic Neuropathy Caused by Focal Venous Engorgement Associated With Deep Vein Thrombosis: A Case Report

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Abstract

A 69-year-old woman complained of severe left leg weakness with paresthesia. Electrodiagnostic tests revealed sciatic neuropathy, and imaging studies showed venous engorgement around the sciatic nerve. After anticoagulant therapy and intensive rehabilitation, the patient's muscle strength improved from 1 to 3 on the Medical Research Council scale. The diagnosis of sciatic neuropathy caused by deep vein thrombosis is extremely rare; however, it should be considered in the differential diagnosis of unilateral lower extremity weakness in patients recovering from intensive care.

Introduction

Sciatic neuropathy is the second most frequent neuropathy in the lower extremities; it can be caused by a number of mechanisms, including acute or chronic nerve entrapment, ischemia, tumor, aneurysm, laceration, toxic injury, vasculitis, hematoma, gluteal muscle contusion, or iatrogenic injury [1,2]. Although vascular causes are the least common, there have been reports of sciatic neuropathy caused by hemangioma, vascular malformation, deep femoral artery aneurysm, traumatic venous varix, or Behçet disease [3-6]. To the best of our knowledge, there has been no previous report of sciatic neuropathy resulting solely from systemic deep vein thrombosis (DVT). In this article, we report a case of sciatic neuropathy associated with vessel engorgement around the sciatic nerve, resulting from disseminated DVT.

Case Presentation

A 69-year-old woman who had undergone chemotherapy for breast cancer experienced uroseptic shock with multiple organ dysfunction and was admitted to the intensive care unit (ICU). The patient's critical care included mechanical ventilation and antibiotic therapy, but no DVT prophylaxis. During her hospital course, the patient had DVTs detected in multiple vessels, including the right popliteal and peroneal veins (muscular branches), left peroneal vein, right great saphenous vein, and right subclavian vein. These were treated with rivaroxaban, a direct, selective, and reversible inhibitor of factor Xa, at the time of diagnosis.

During her ICU stay, the patient complained of profound weakness in both lower extremities and pain in her left lower extremity. She also noted paresthesia below her knee, particularly in the sole of her left foot. Because of these complaints and her poor overall condition, the physiatry department was consulted. Her initial neurologic examination was limited due to her extremely poor overall condition. Initially the patient was believed to have critical illness neuropathy (CIN) or critical illness myopathy (CIM), given her lack of a definitive focal neurologic deficit and in the setting of a symmetric weakness that affected the proximal muscles more than distal muscles.

The patient was transferred to the rehabilitation unit because of her continued weakness for 1 month even after her medical condition stabilized. On initial manual muscle examination at the rehabilitation unit, the patient was found to have grade 1 of 5 strength on the Medical Research Council (MRC) muscle strength scale in her left ankle dorsiflexion and plantar flexion. Because asymmetric weakness is an atypical presentation for CIN or CIM, we eliminated CIN or CIM from the differential diagnosis and decided to perform an electrodiagnostic study.

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Electrodiagnostic tests performed approximately 1 month after the patient left the ICU revealed left sciatic neuropathy with moderate to severe partial axonal involvement, involving the tibial branch of the nerve more severely than the peroneal branch (Tables 1 and 2). There was no evidence of generalized peripheral polyneuropathy or myopathy. and critical illness neuropathy and myopathy were also excluded. The findings of abnormal recruitment of the vastus medialis and the tibialis anterior were believed to be volitionally inhibited and not representative of neuropathic disease. Magnetic resonance imaging of the pelvis revealed prominent perineural and intraneural veins adjacent to the sciatic nerve, suggesting a vascular malformation or varix (Figure 1). Three-dimensional computed tomographic angiography of the left leg showed focal enlargement and enhancement of the veins around the sciatic nerve posterior to the hip joint capsule, with a mildly engorged venous structure between the inferior gemellus and quadratus femoris muscles (Figure 2). An abdominal computed tomography (CT) scan taken when the patient first presented with uroseptic shock showed no vascular abnormalities near the left sciatic nerve, whereas a later scan taken during the patient's time in the ICU showed newly developed venous engorgement near the nerve (images not shown). On the basis of the electrodiagnostic and imaging data, the patient was diagnosed with sciatic neuropathy, presumably due to vascular engorgement. The vascular engorgement was noted to be small, and difficult to access surgically. Discussions with a vascular surgeon revealed considerable uncertainty regarding the results of surgical management. These factors, combined with the lack of progression of her neurologic deficits, resulted in a decision to remain conservative and to treat with anticoagulation therapy. In addition, gabapentin and tramadol were prescribed for her neuropathic pain. Her rehabilitation involved gait training with a walker, quad cane, and left ankle orthosis. She was discharged after spending 1 month in the rehabilitation unit.

Three months after discharge, the patient visited the outpatient rehabilitation clinic for a follow-up appointment. She was now able to walk without an orthosis. Her ankle dorsiflexion was grade 4 on the MRC scale, and her plantar flexion was grade 3. CT angiography showed reduced focal enlargement and enhancement of the veins around the sciatic nerve, and there was no evidence of DVT in the lower extremities. The patient continued to return for follow-up every 3 months, and her condition gradually improved. She was able to discontinue anticoagulant therapy 6 months after discharge. Nine months after discharge, she underwent follow-up electrodiagnostic tests. Needle electromyography showed improved function of the peroneus longus and biceps femoris muscles, but there were no other changes.

Discussion

Focal sciatic neuropathy is the second most common mononeuropathy in the legs after peroneal neuropathy and may be caused by a variety of conditions; however, the cause is very rarely vascular. The most commonly reported vascular cause of focal sciatic neuropathy is arterial or venous thrombosis,

Table 1
Nerve conduction study results

Motor Nerve Conduction Studies

Nerve	Side	Record	Baseline to Peak Amplitude (mV) Distal	Onset Latency (ms) Distal	Nerve Conduction Velocity (m/s)
Peroneal	R	EDB	2.5	4.2	38.6
Peroneal	L	EDB	1.1	4.6	39.4
Peroneal	R	TA	5.9	3.3	61.1
Peroneal	L	TA	2.9	2.4	35.3
Tibial	R	AH	5.7	5.3	41
Tibial	L	AH	No response		

F-Wave Latencies

Nerve	Side	Record	Latency (ms)
Tibial	R	AH	No response
Tibial	L	AH	No response

Sensory Nerve Conduction Studies

Nerve	Side	Record	Baseline to Peak Amplitude (mV)	Onset Latency (ms)	Peak-to-Peak Amplitude (mV)
Sural	R	Ankle	12.3	2.8	9.4
Sural	L	Ankle	6.7	2.7	3.7
Superficial peroneal	R	Foot	6.7	2.8	6
Superficial peroneal	L	Foot	5.5	3.9	4.8

R = right; L = left; EDB = extensor digitorum brevis; TA = tibialis anterior; AH = abductor hallucis.